CARDIAC ARRHYTHMIAS

Cardiac arrhythmias

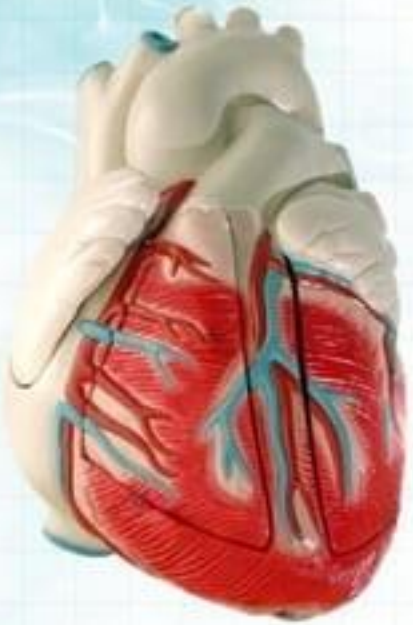
- An abnormality of the cardiac rhythm is called a cardiac arrhythmia. Arrhythmias may cause sudden death, syncope, heart failure, dizziness, palpitations or no symptoms at all. There are two main types of arrhythmia:
  1. bradycardia: the heart rate is slow (< 60 b.p.m.)
  2. tachycardia: the heart rate is fast (> 100 b.p.m.).

Cardiac arrhythmias

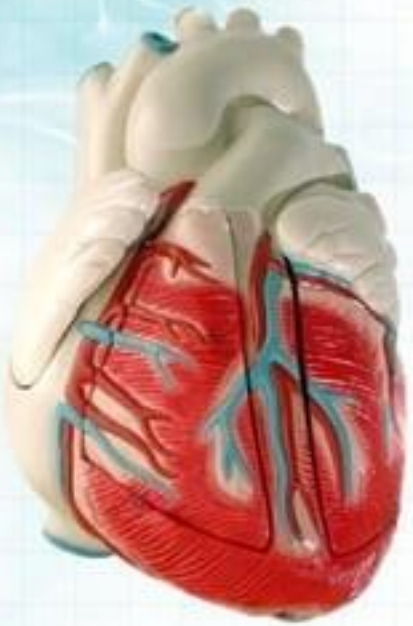
- Tachycardias are more symptomatic when the arrhythmia is fast and sustained. Tachycardias are subdivided into supraventricular tachycardias, which arise from the atrium or the atrioventricular junction, and ventricular tachycardias, which arise from the ventricles.

SINUS RYTHM

- The normal cardiac pacemaker is the sinus node
- its rate of discharge is controlled by the autonomic nervous system; sympathetic system increases heart rate, while parasympathetic system decrease it. Normal sinus rhythm is characterized by P waves that are upright in leads I, and II of the ECG, but inverted in the cavity leads AVR and V1.



Sinus arrhythmia

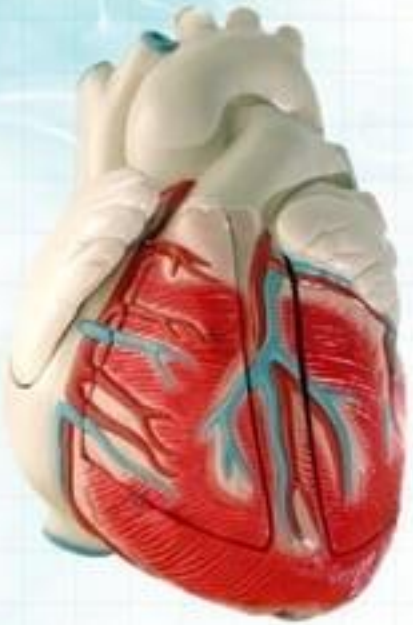


- Fluctuations of autonomic tone result in phasic changes of the sinus discharge rate. During inspiration, parasympathetic tone falls and the heart rate quickens, and on expiration the heart rate falls. This variation is normal, particularly in children and young adults. Typically sinus arrhythmia results in a regularly irregular pulse.

Cardiac arrhythmias

Arrhythmia Formation

- Arrhythmias can arise from problems in the:
  - Sinus node
  - Atrial cells
  - AV junction
  - Ventricular cells
- Arrhythmias caused by 2 main mechanism:
  - Automaticity
  - Re-entry.

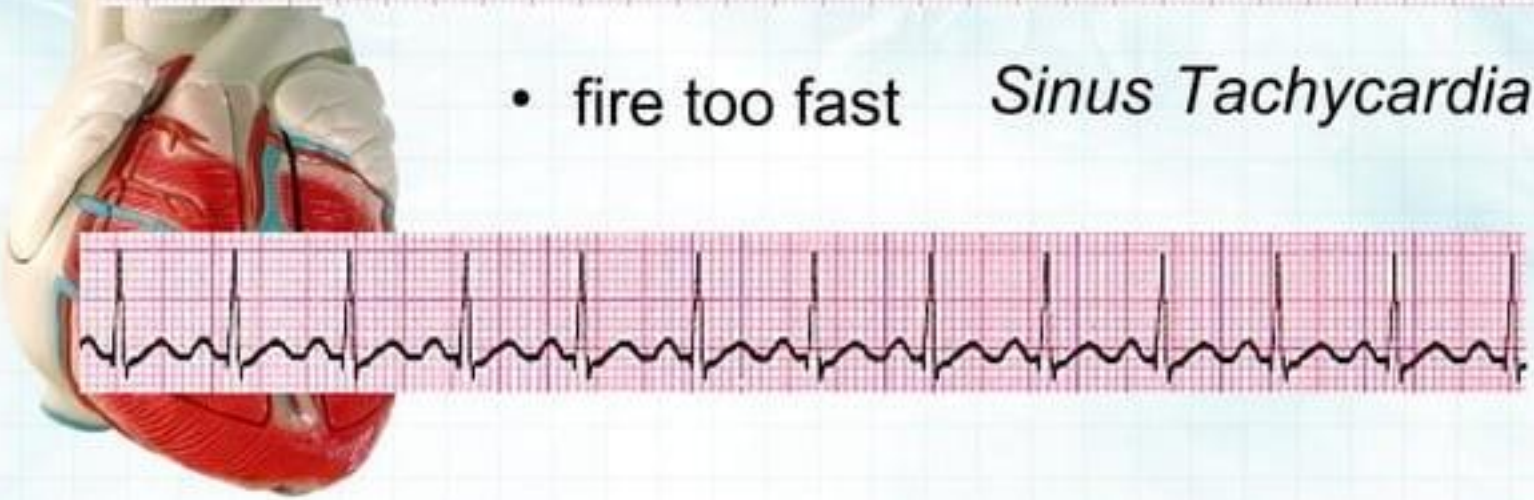


SA Node Problems

The SA Node can:

- fire too slow *Sinus Bradycardia*

- fire too fast *Sinus Tachycardia*



Cardiac arrhythmias

Atrial Cell Problems

Atrial cells can:

- fire occasionally from a focus

Premature Atrial Contractions (PACs)

- Fire continuously due to a looping re-entrant circuit

Atrial Flutter

Atrial Cell Problems

Atrial cells can also:

- **fire continuously from multiple foci**

Atrial Fibrillation

or

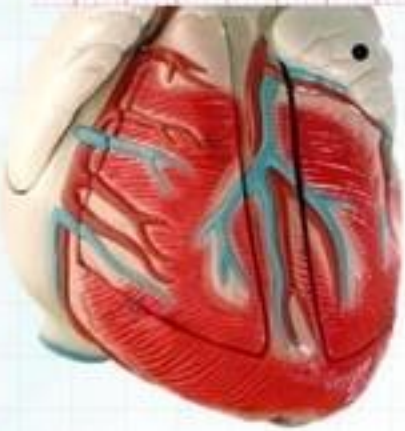
- **fire continuously due to multiple micro re-entrant "wavelets"**

Atrial Fibrillation

AV Junctional Problems

The AV junction can:

- fire continuously due to a looping re-entrant circuit — Paroxysmal Supraventricular Tachycardia



- block impulses coming from the SA Node — AV Junctional Blocks

Ventricular Cell Problems

Cardiac arrhythmias

Ventricular cells can:

- fire occasionally from 1 or more foci

Premature Ventricular Contractions (PVCs)

- fire continuously from multiple foci

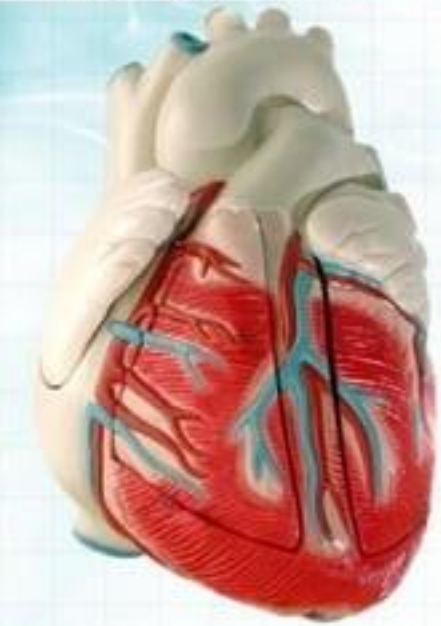
Ventricular Fibrillation

- fire continuously due to a looping re-entrant circuit

Ventricular Tachycardia

Sinus Bradycardia

Cardiac arrhythmias

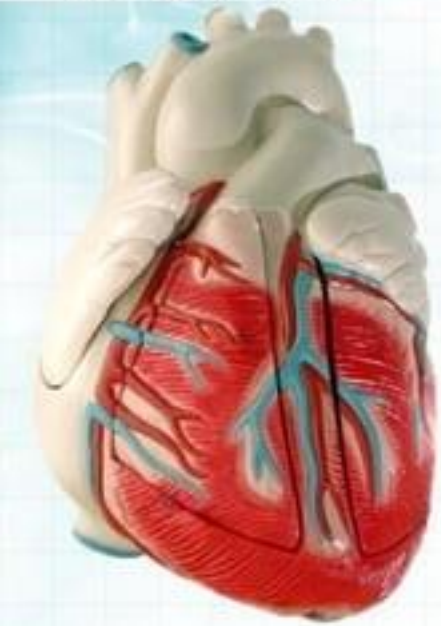


- A sinus rate of less than 60 b.p.m. during the day or less than 50 .m. at night is known as sinus bradycardia.
- CAUSES

Physiological

- In athletes
- During sleep

Cardiac arrhythmias



Pathological

Extrinsic causes

1. Hypothermia, hypothyroidism, cholestatic jaundice and raised intracranial pressure.
2. Drug therapy with beta-blockers, digitalis and other antiarrhythmic drugs.
3. Neurally mediated syndromes

Cardiac arrhythmias

Intrinsic causes

1. Acute ischaemia and infarction of the sinus node (as a complication of acute myocardial infarction).
2. Chronic degenerative changes such as fibrosis of the atrium and sinus node (sick sinus syndrome).

Sick sinus syndrome

Cardiac arrhythmias

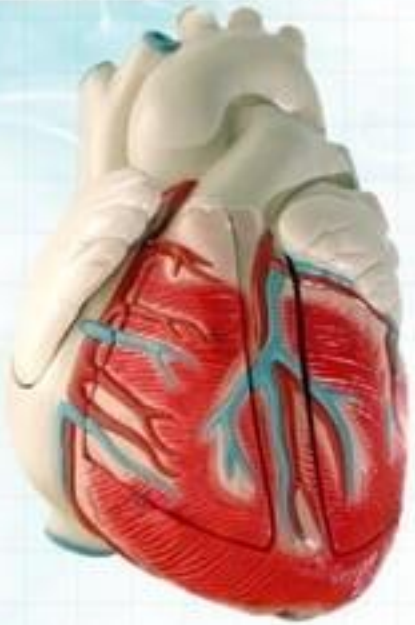
Sick sinus syndrome or *sinoatrial disease* is usually caused by idiopathic fibrosis of the sinus node. Other causes of fibrosis such as ischemic heart disease, cardiomyopathy or myocarditis can also cause the syndrome. Patients present episodes of sinus bradycardia, sinus arrest, paroxysmal superaventricular tachycardia or tachy-brady syndrome.

CLINICAL FEATURES

Cardiac arrhythmias

- Pt may be asymptomatic
- Severe bradycardia may cause weakness, confusion & syncope
- Atrial & ventricular ectopics are more apparent to occur with slow sinus rate.

Cardiac arrhythmias



TREATMENT

- Identification & treatment of the cause
- Acute symptomatic bradycardia responds to atropine o.6 mg i.v.
- Temporary pacemaker in symptomatic bradycardia if there is reversible cause
- Permenant pacemaker in symptomatic irreversible cause

Cardiac arrhythmias

Sinus tachycardia

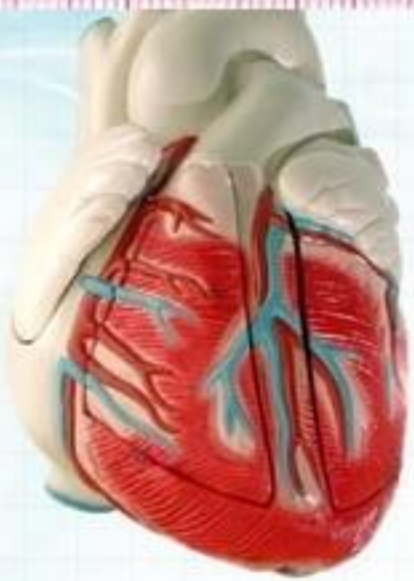
- Resting sinus rate of more than 100 b.p.m. is known as sinus tachycardia.

Causes

Non cardiac

Acute

1. Fever
2. Hypovolemia
3. Pain
4. Infection
5. Exercise
6. Emotion/anxiety



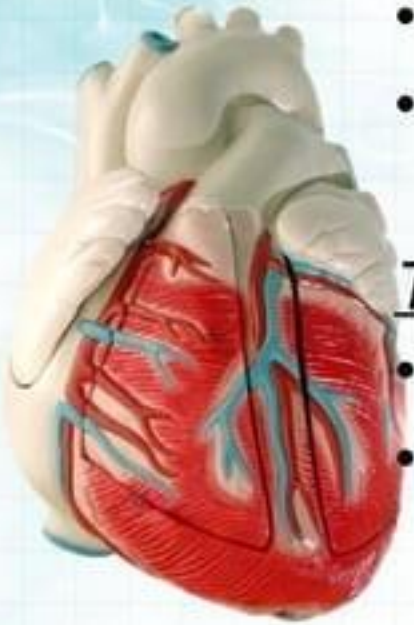
Cardiac arrhythmias

Cardiac

HF with compensatoy tachycardia

CLINICAL FEATURES

- Onset & termination are gradual
- not exceed 160 b.p.m.
- Pt complain of palpitation

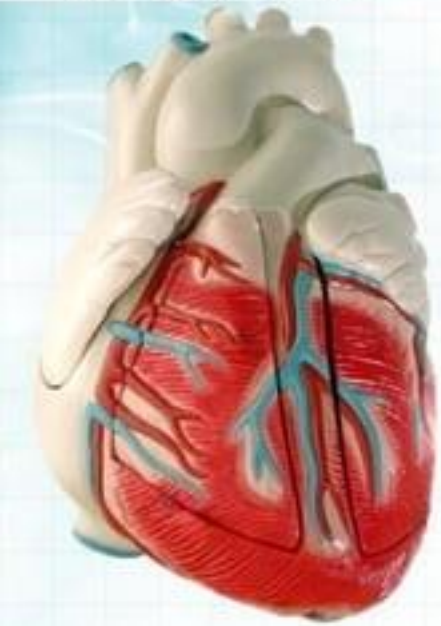


TREATMENT

- Treatment of the cause
- Symptomatic sinus tachycardia can be reduced with beta-blocker and/or Verapamil

HEART BLOCK

Cardiac arrhythmias



Heart block or conduction block may occur at any level in the conducting system. Block in either the AV node or the His bundle results in Atrioventricular (AV) block, whereas block lower in the conduction system produces bundle branch block.

Atrioventricular block

Cardiac arrhythmias

There are three forms:

First-degree AV block

- This is simple prolongation of the PR interval to more than 0.22 s. Every atrial depolarization is followed by conduction to the ventricles but with delay.
- Delay of conduction may be within atrium, AV node, bundle of his or bundle branches.

Second-degree AV block

This occurs when some P waves conduct and others do not. There are several forms

There are three types:-

Cardiac arrhythmias

Mobitz I block (Wenckebach block phenomenon) is progressive PR interval prolongation until a P wave fails to conduct. The PR interval before the blocked P wave is much longer than the PR interval after the blocked P wave.

Cardiac arrhythmias

2. ***Mobitz II block*** occurs when a dropped QRS complex is not preceded by progressive PR interval prolongation.

Cardiac arrhythmias

3 2 : 1 or 3 : 1 (advanced) block

- It may present as either type I or type II AV block in which there are 2 p waves or 3 to each QRS complex. If PR interval is prolonged and QRS is narrow then it is type I. if PR normal and QRS is wide then it is type II 2nd degree AV block.

Causes of AV block

- Ischemia of the AV node or AV bundle fibers
- Compression of the AV bundle by scar tissue of calcified portions of the heart
- Inflammation of the AV node
- Extreme stimulation of parasympathetic system

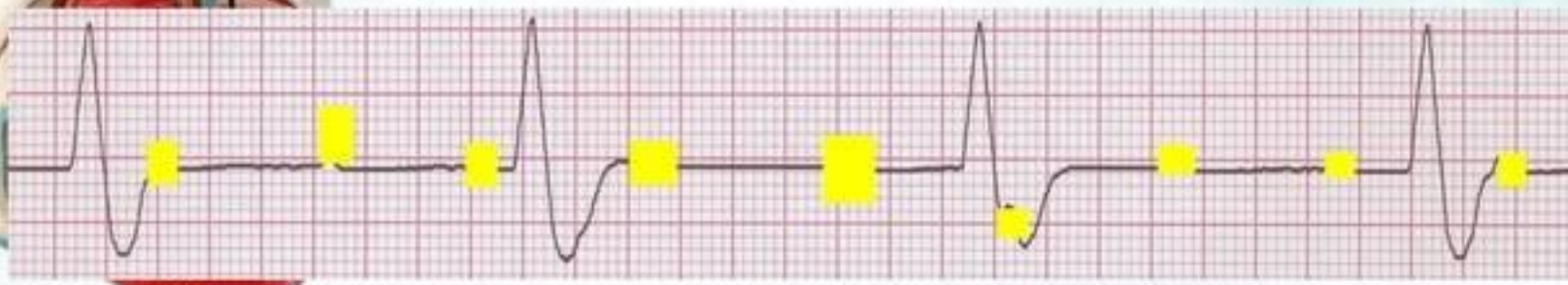
(for example carotid sinus syndrome)

Cardiac arrhythmias

Third-degree (complete) AV block

Cardiac arrhythmias

- Complete heart block occurs when all atrial activity fails to conduct to the ventricles. In patients with complete heart block the etiology needs to be established. Cardiac action is maintained by spontaneous escape rhythm.



Etiology of complete heart block

Cardiac arrhythmias

1. **Congenital**
2. **Idiopathic fibrosis**
3. **Ischaemic heart disease**
4. Infiltrations (e.g. amyloidosis, sarcoidosis, neoplasia)
5. **Cardiac surgery**
6. **Drug-induced**e.g. digoxin, amiodarone
7. **Infections:** Endocarditis, Lyme disease & Chagas' disease
8. **Connective tissue diseases**e.g. SLE, rheumatoid arthritis

Cardiac arrhythmias

SYMTOMS OF HEART BLOCK

symptom develop due to bradycardia & loss of AV node synchrony

- Exercise intolerance
- Easy fatigability
- Dyspnea on exertion
- Dizzy spills
- Near syncope & frank syncope

Cardiac arrhythmias

Treatment

Heart block complicate acute MI

- Acute inferior wall MI is often complicated by transient AV block which may respond to atropine 0.6 mg i/v repeated as necessary, if it fail temporary pacemaker should be inserted. In majority AV block will resolve within 7-10 days. Pt may develop heart failure or hypotension due to slow heart rate in already damaged heart requiring pacemaker.

Cardiac arrhythmias

- Mobitz II or complete heart block complicating anterior wall MI is usually a sign of extensive myocardial damage and carries a poor prognosis, asystole often occurs and temporary pacemaker should be inserted prophylactically as soon ass possible.
- If the pt presents with asystole, atropine 0.6 mg i/v repeated as necessary and isopronaline1-5mg in 500ml dextrose 5% infusion I/V at minimum rate until temporary pacemaker can be inserted

Cardiac arrhythmias

Chronic heart block

- Asymptomatic 1st degree or Mobitz type I 2nd degree AV block requires no treatment.
- Permanent pacemaker in pt with symptomatic bradycardia with complicating AV block.
- Permanent pacemaker in pt with asymptomatic Mobitz II 2nd or complete heart block.

Cardiac arrhythmias

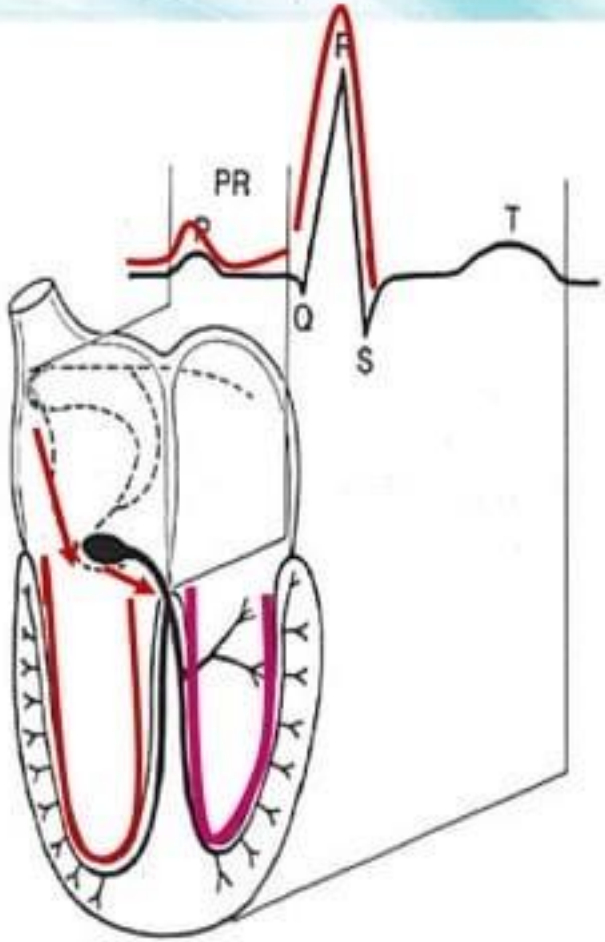
Bundle branch block

- The His bundle gives rise to the right and left bundle branches. The left bundle subdivides into the anterior and posterior divisions of the left bundle. Various conduction disturbances can occur.

Normal Impulse Conduction

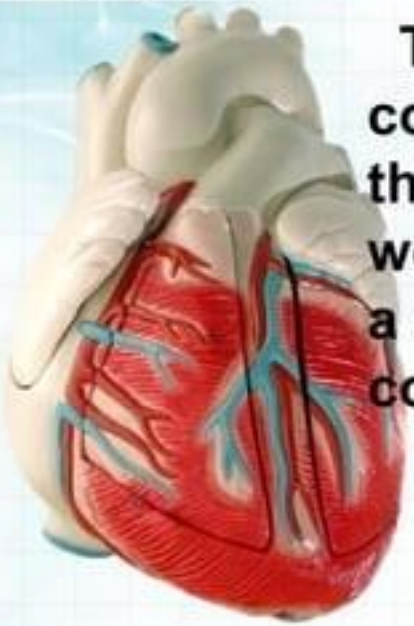
Cardiac arrhythmias

- Sinoatrial node
- AV node
- Bundle of His
- **Bundle Branches**
- Purkinje fibers

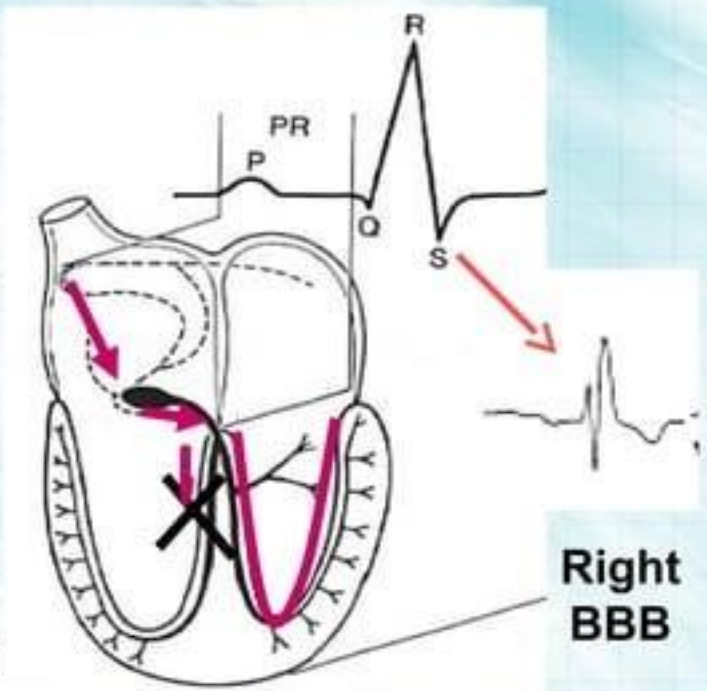


Cardiac arrhythmias

So, depolarization of the Bundle Branches and Purkinje fibers are seen as the QRS complex on the ECG.



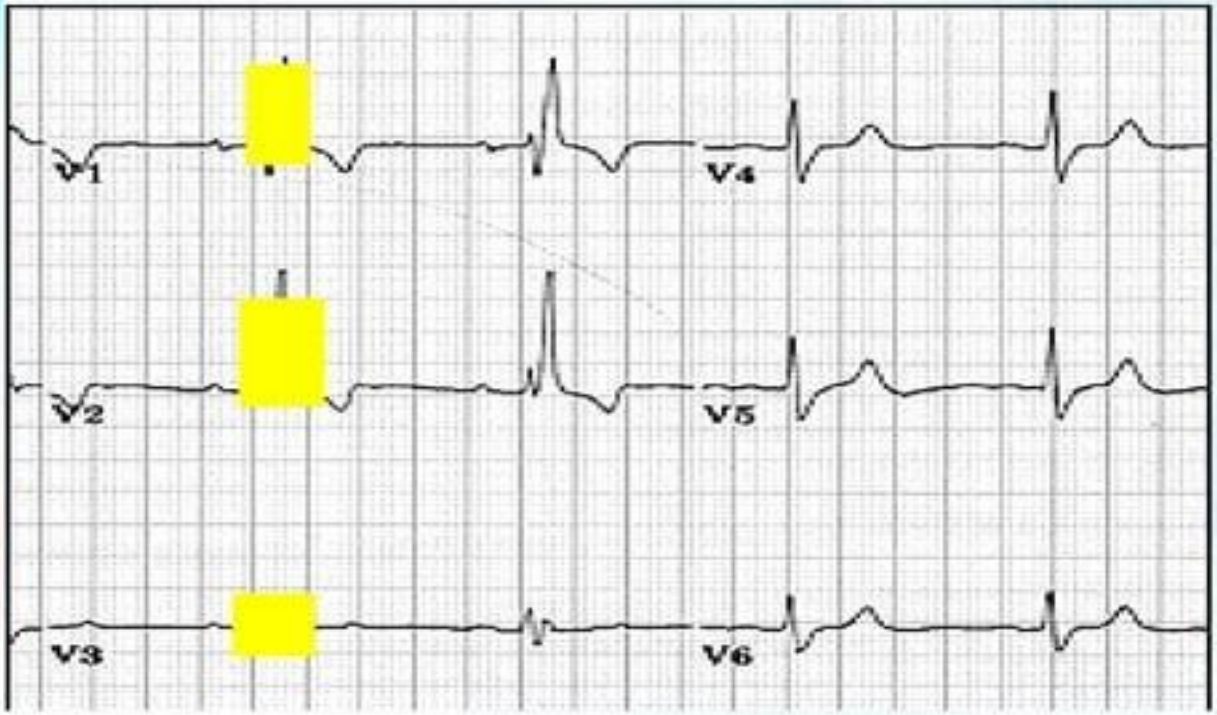
Therefore, a conduction block of the Bundle Branches would be reflected as a change in the QRS complex.



With Bundle Branch Blocks you will see two changes on the ECG.

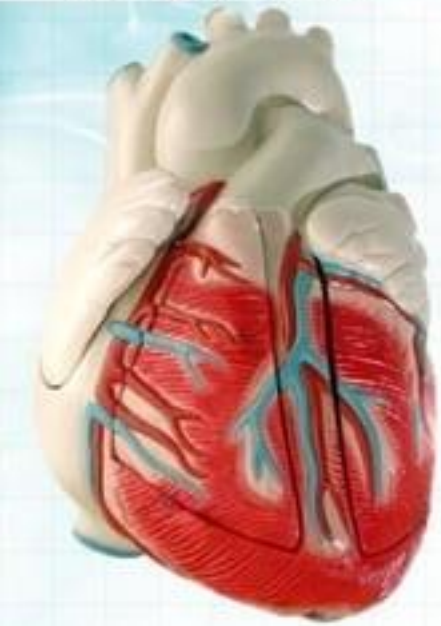
1. QRS complex widens (> 0.12 sec).
2. QRS morphology changes (varies depending on ECG lead, and if it is a right vs. left bundle branch block).

Cardiac arrhythmias

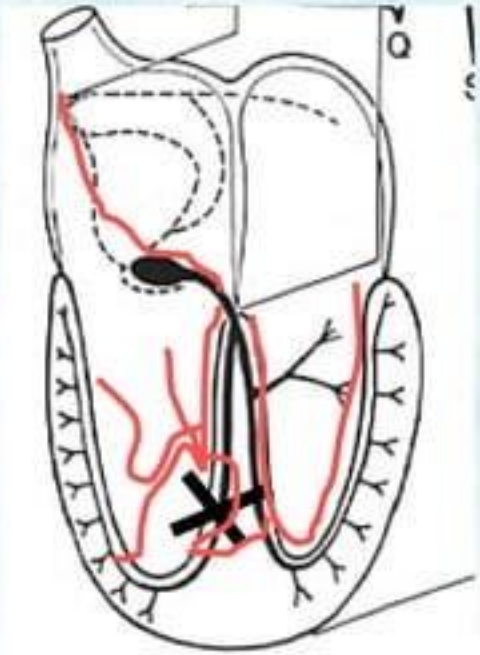


Why does the QRS complex widen?

Cardiac arrhythmias



When the conduction pathway is blocked it will take longer for the electrical signal to pass throughout the ventricles.

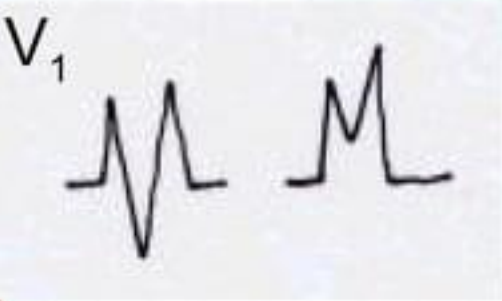


Right Bundle Branch Blocks

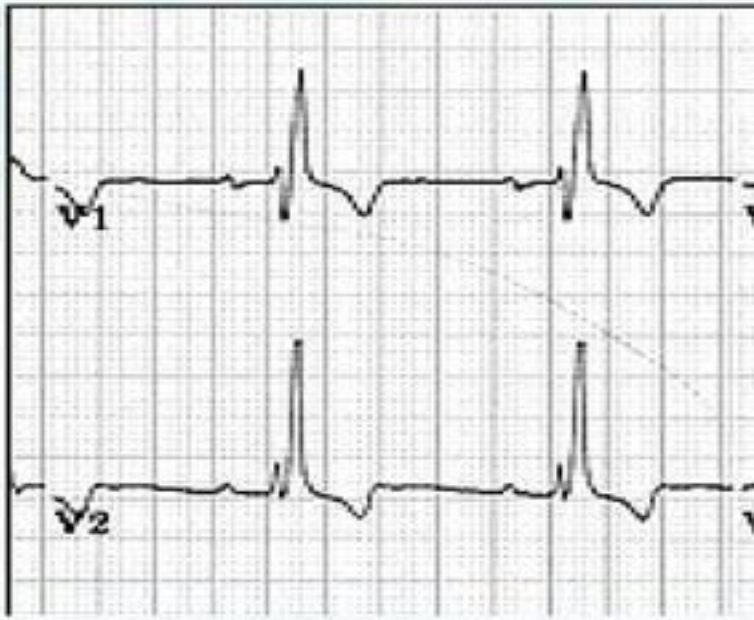
- **What QRS morphology is characteristic?**

  For RBBB the wide QRS complex assumes a unique, virtually diagnostic shape in those leads overlying the right ventricle (V1 and V2).

Cardiac arrhythmias



Rabit ear (M)



Causes of right bundle branch block

Cardiac arrhythmias

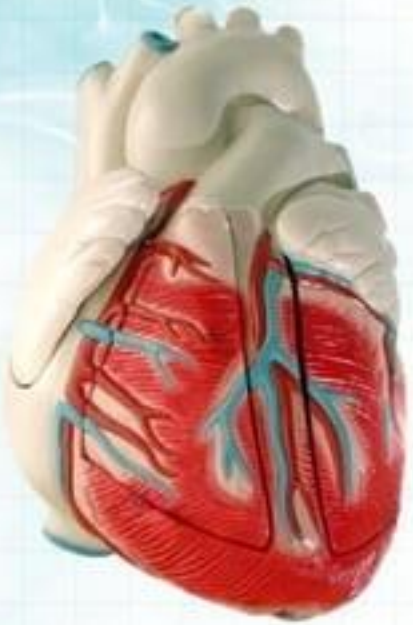
It is a normal finding in 1% of young adults and 5% of elderly adults

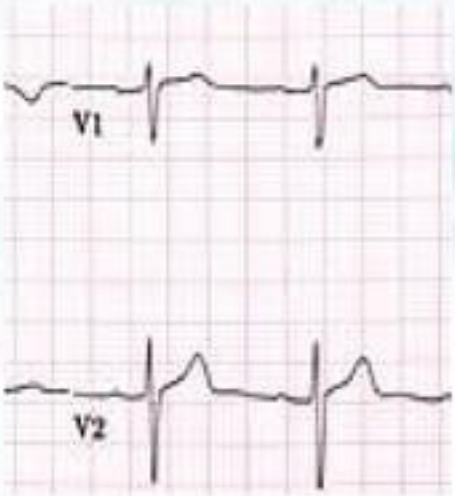
- **Congenital heart disease**
- **Pulmonary disease**
- Acute myocardial infarction
- Cardiomyopathy
- Conduction system fibrosis

Left Bundle Branch Blocks

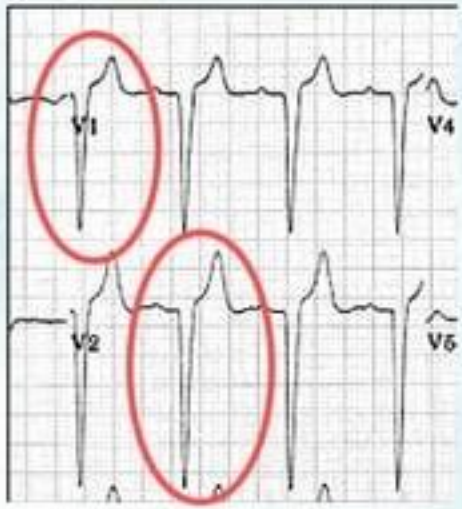
What QRS morphology is characteristic?

For LBBB the wide QRS complex assumes a characteristic change in shape in those leads opposite the left ventricle (right ventricular leads - V_1 and V_2).





Normal



Broad, deep S waves

. *Causes of left bundle branch block*

Cardiac arrhythmias

- **Left ventricular outflow obstruction**
- Aortic stenosis
- Hypertension
- **Coronary artery disease**
- Acute myocardial infarction
- Severe coronary disease (two- to three-vessel disease)

Cardiac arrhythmias

- In each of them there are wide QRS
- Broad notched R wave in leads V1 & V2 in RBBB but in leads V5, V6, I & aVL in LBBB.
- Wide deep S waves in leads V5 & V6 in RBBB. But in leads V1 & V2 in LBBB.
- LBBB it indicate more severe disease
- diagnosis of LVH or MI is difficult in the presence of LBBB.

Cardiac arrhythmias

- Right bundle branch block alone does not alter the electrical axis of the heart. Axis deviations signify right ventricular hypertrophy (RV overload) or coexistent fascicular block. The combination of right bundle branch block with left axis deviation is associated with ostium primum atrial septal defects.

PATHOLOGICAL TACHYCARDIA

ECTOPIC RHYTHM

Cardiac arrhythmias

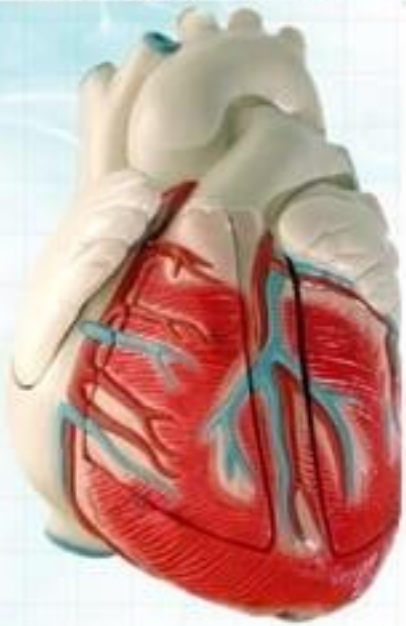
Impulse arises somewhere other than SA node.

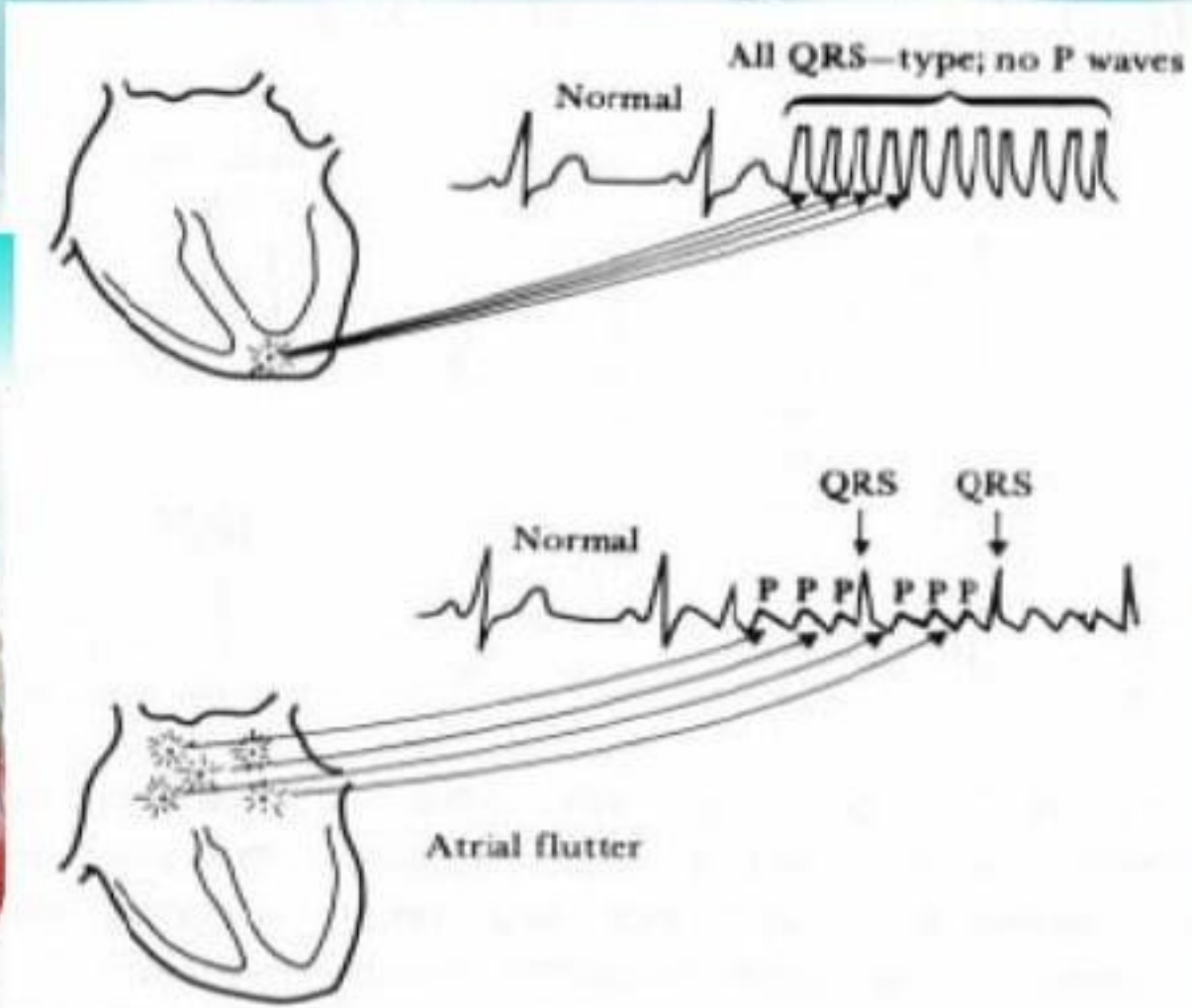
May arise from:-

- Supraventricular (including, AV node or other AV junctional tissue).
- Ventricular in the ventricle

Ventricular and atrial tachycardia

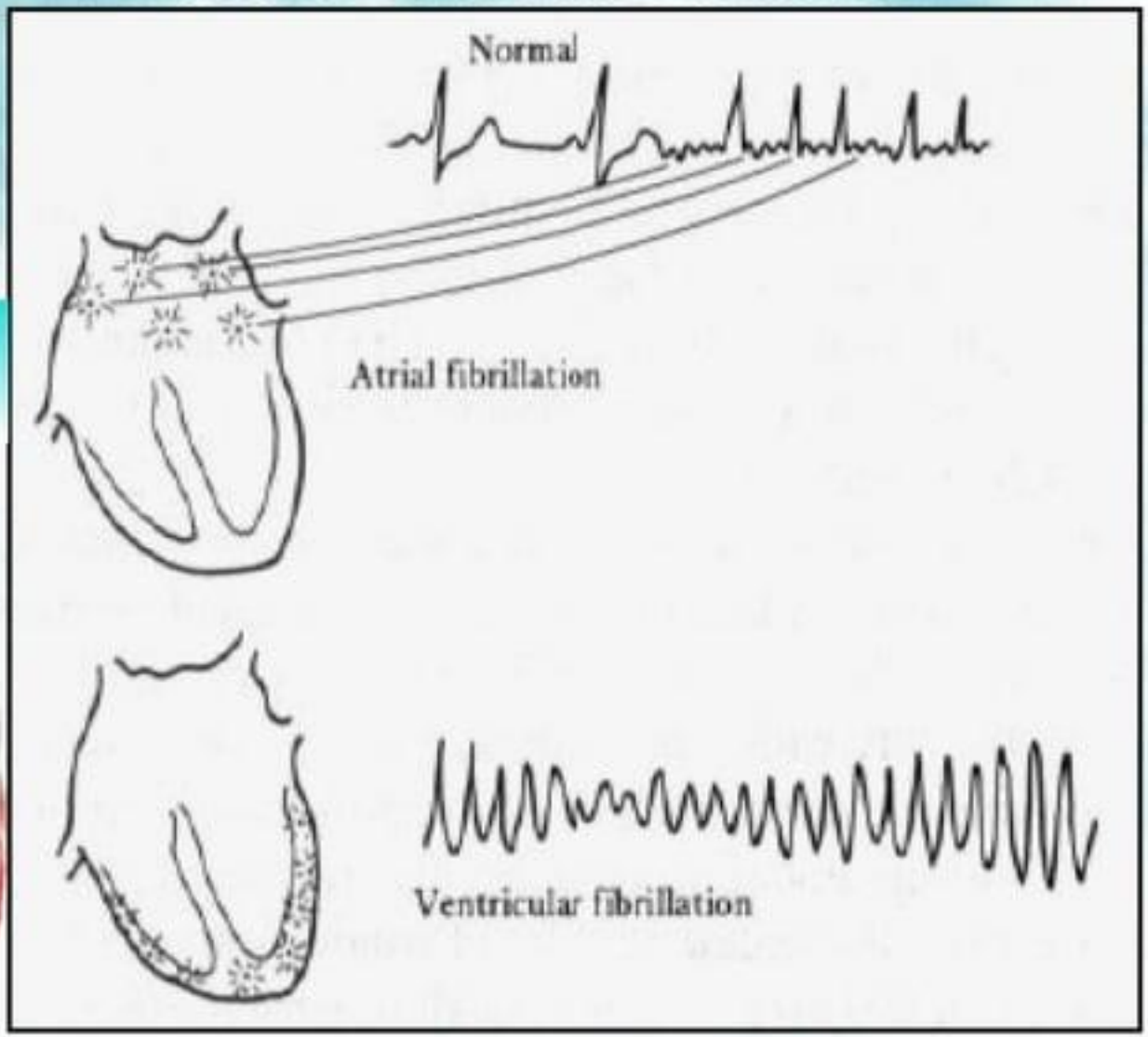
Cardiac arrhythmias





Atrial and ventricular fibrillation

Cardiac arrhythmias



supraventricular ectopic rhythm

Ectopic beats

Cardiac arrhythmias

- Mostly asymptomatic
- Can give sensation of an extra or extra beat
- ECG shows a premature beat with a normal QRS complex.

supraventricular tachycardia (SVT)

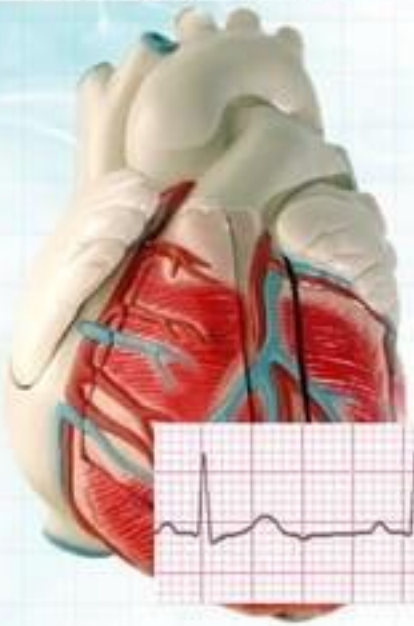
Cardiac arrhythmias

- This is a tachycardia occurring in episodes with rate of 140-240, as a result of re-entry or rapidly firing ectopic focus in the atria or AV node. It may last from few seconds to many hours (if untreated)
- *Predisposing factors:*
  - Anxiety
  - Excess tobacco or coffee
  - Hyperthyroidism
  - Exertion
  - Alcohol

Clinical features

- Pt feels that has suddenly start to beat fast (palpitation).
- Fainting, breathlessness, dizziness, neck pulsation, central chest pain, and weakness
- polyuria is some time a feature.

Cardiac arrhythmias

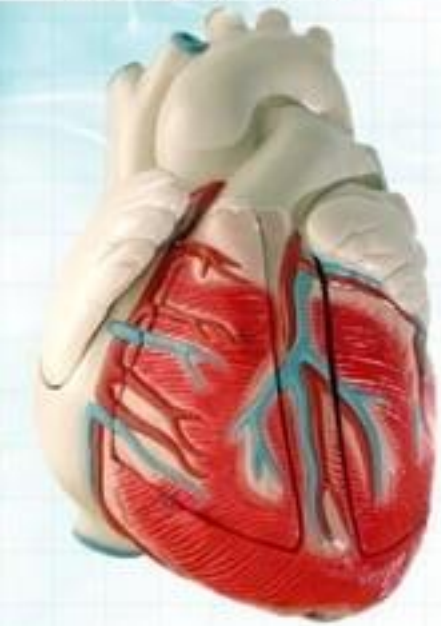


ECG shows tachycardia with normal QRS complex

Management

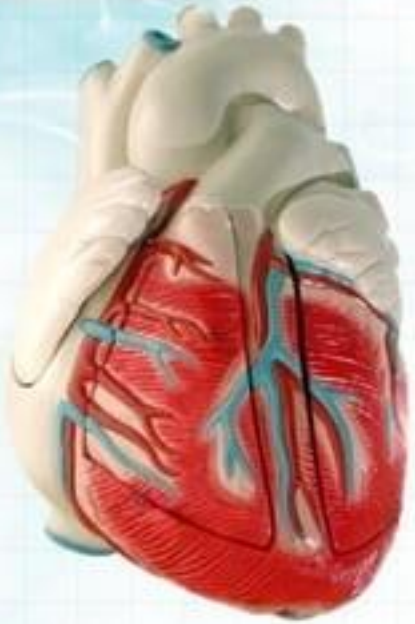
Acute management

Cardiac arrhythmias



In the absence of heart disease, serious side effects are rare, and most attacks break spontaneously. Terminate attack if cardiac failure, syncope or anginal pain develops or if there underlying cardiac or coronary disease.

Cardiac arrhythmias



vagotonic maneuvers

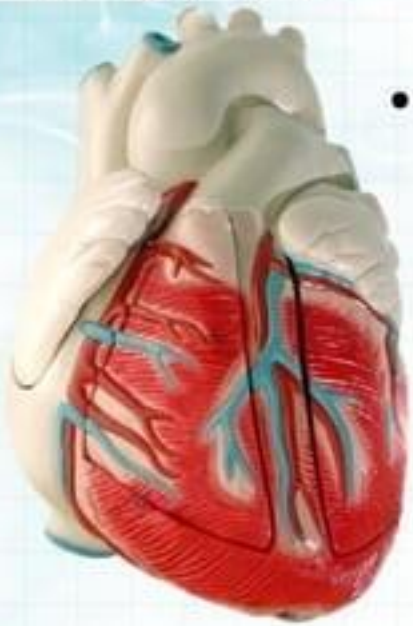
- Carotid sinus message
- Valsalva maneuver
- facial immersion in cold water
- Pressure on eyes
- Self inducing vomiting
- Breath holding
- Lowering the head between the knees

drugs

- If physical manoeuvres have not been successful,
- intravenous adenosine 6mg bolus, if no response within 1-2 minutes 2nd & 3rd 12mg should be given. SE bronchospasm, flushing & chest pain.
- verapamil 5-10 mg i.v. over 5-10 minutes Maintenance 2.5–10 mg/h.
- Beta blokers ismolol 500microg/mkg within one minute foolwed by 25-200microg/min.

Cardiac arrhythmias

Cardiac arrhythmias



- Digoxin is also effective but it take longer time to act
- Beta blocker and digoxin are less commonly used
- Amiodarone is not required for termination of attack, it is used for prevention of occurrence.
- if pt hymodynamically unstable or if adenosine or veramapil are contraindicated or in effective. Synchronized DC cardioversion should be performed.

Long-term management

Cardiac arrhythmias

Verapamil, diltiazem, and beta-blockers have proven effective in 60-80% of patients.

If drug therapy is not effective then perform electrophysiological evaluation and perform catheter mediated ablation.

Atrial tachyarrhythmias

- Atrial tachyarrhythmias including atrial fibrillation, atrial flutter, atrial tachycardia and atrial ectopic beats all arise from the atrial myocardium. They share common etiologies.

Cardiac arrhythmias

CAUSES

Cardiac

- Hypertension
- Congestive heart failure
- Coronary artery disease and myocardial infarction
- Cardiomyopathy: dilated, hypertrophic
- Myocarditis and pericarditis
- Wolff-Parkinson-White syndrome
- Sick sinus syndrome
- Cardiac tumours
- Cardiac surgery

Cardiac arrhythmias

Cardiac arrhythmias

Non-cardiac

- Thyrotoxicosis
- Phaeochromocytoma
- pneumonia, COPD
- pulmonary embolism
- hypokalaemia
- Alcohol abuse
- Caffeine & smoking
- Idiopathic

ATRIAL FLUTTER

Cardiac arrhythmias

- It is a condition in which atrial rate typically between 250 and 350 beats per minute.
- *The ECG* shows regular saw tooth-like atrial flutter waves (F waves) between QRS complexes

Clinical features:

- Palpitation
- Reduced COP leads to fatigue, weakness, coolness of the skin & light headiness. In adequate coronary perfusion may cause angina, inadequate cerebral perfusion may lead to dizziness or syncope.

Management

- DC cardioversion at < 50J as initial treatment of choice
- Procainamide 15 mg/kg over 60 min maintainace1–4 mg/min can be given for cardioversion
- Verapamil to slow ventricular response
- Digitalis with beta-blocker or calcium channel blocker may be given to slow ventricular rate if electrical or pharmacological cardioversion is not possibl.
- Amiodarone 15 mg/min for 10 min, 1 mg/min for 6 h to restore sinus rhythm and prevent reccurence of atrial flutter
- .radiofrequency catheter ablation.

Cardiac arrhythmias

Atrial fibrillation

Cardiac arrhythmias

- Atrial fibrillation is a continuous, rapid (300-600 per minute) activation of the atria. The atria beats rapidly and ineffectively , the ventricle\s respond at irregular intervals giving the characteristic irregularly irregular pulse.
- **ECG**
  - Absent P wave
  - Fine oscillation of baseline (fibrillation waves).
  - QRS rhythm is rapid & irregular.

CLINICAL FEATURES

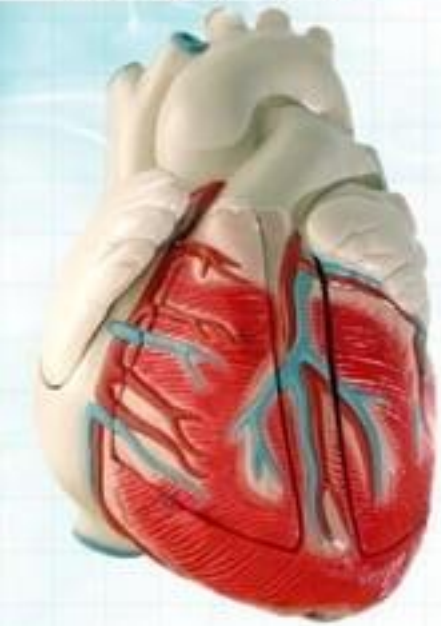
1. Palpitation
2. if ventricular response is rapid, COP may fall resulting in:
   - Symptoms of pulmonary congestion (dyspnea, orthopnea & PND)
   - Symptoms of inadequate peripheral perfusion (angina, dizziness& syncope)
1. Systemic embolism (stroke, leg pain & abdominal pain)

Cardiac arrhythmias

Management

- When atrial fibrillation is due to an acute precipitating event such as alcohol toxicity, chest infection or hyperthyroidism, the provoking cause should be treated. Strategies for the acute management of AF are ventricular rate control or cardioversion (± anticoagulation). Ventricular rate control is achieved by drugs which block the AV node. while the cardioversion is achieved electrically by DC shock or medically either by intravenous infusion of an anti-arrhythmic drug such as a class Ic or a class III agent or by taking an oral agent previously tested in hospital and found to be safe in a particular patient ('pill-in-pocket' approach).

Cardiac arrhythmias



Cardiac arrhythmias

Electrical DC cardioversion

- If pt is unstable due to fast ventricular rate & presents in shock, severe hypotension, pulmonary edema or ongoing MI. the risk o fthromboembolism is high if atrial fibrillation persist more than 48 hrs.
- Conversion to sinus rhythm can be achieved by electrical DC cardioversion 200 J, then 2 × 360 J in about 80% of patients

Rate control

- In less unstable pt or those at high risk of thromboembolism due to cardioversion
- Rate control is usually achieved by a combination of *Digoxin, beta-blockers* or *calcium-channel blockers* (*Verapamil* or *diltiazem*). The ventricular rate response is generally considered controlled if the heart rate is between 60 and 80 beats per minute at rest and 90 and 115 beats per minute during moderate exercise

Cardiac arrhythmias

Cardiac arrhythmias

- If rate control is unsuccessful an duration of atrial fibrillation is more than 2-3 days then perform transoesophageal echocardiography to look for atrial thrombus, if there is no thrombus then cardiovert the pt, in case of presence of thrombus give anticoagulation for 4 wks before & 4 wks after cardioversion.

Cardiac arrhythmias

Two strategies are available for the long-term management of atrial fibrillation:

1. 'rhythm control' (antiarrhythmic drugs plus DC cardioversion *plus warfarin*)
2. 'rate control' (AV nodal slowing agents *plus warfarin*).

Elective cardioversion

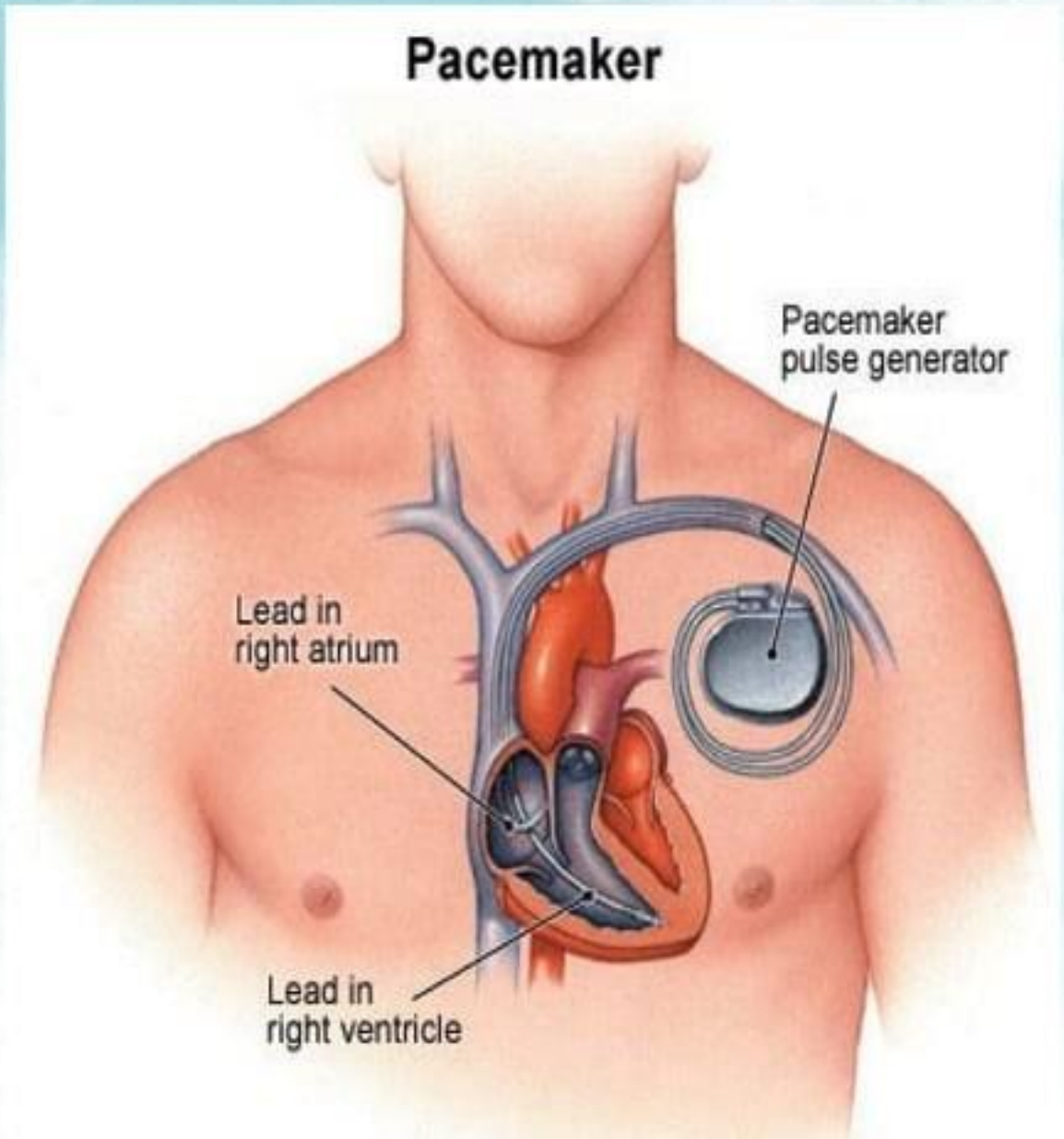
First anticoagulate the pt for 4 wks.

Pharmacological cardioversion by using amiodarone 300-400mg twice daily for 2-4wks then 200mg daily . Anticoagulation should be continued.

Radiofrequency VA node ablation & insertion of permanent pacemaker should be used in resistant cases with no drug works

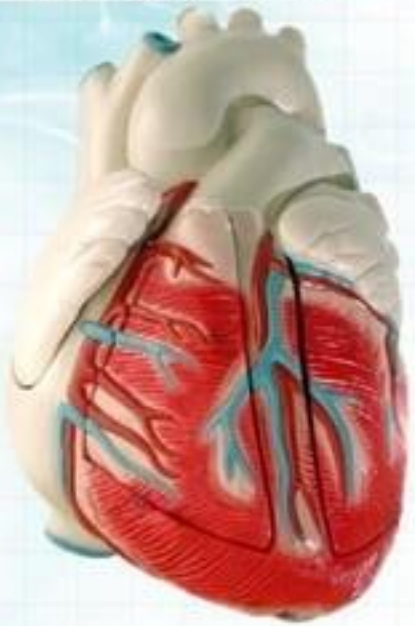
Cardiac arrhythmias

Cardiac arrhythmias



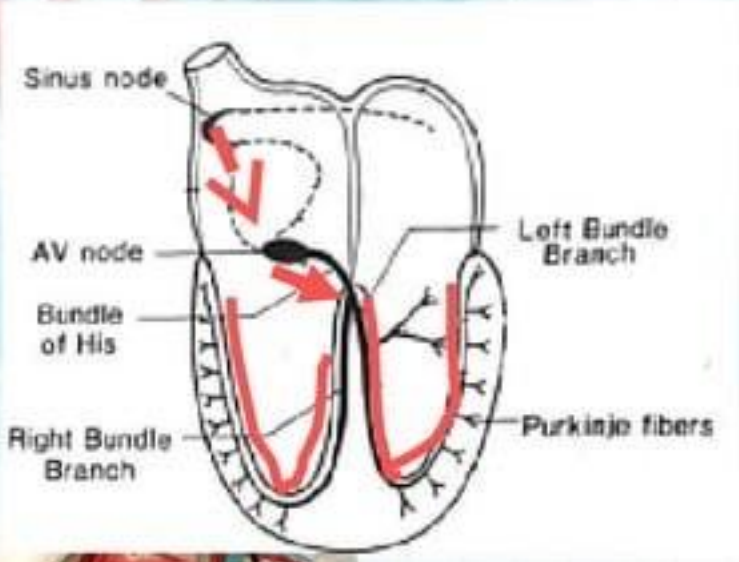
Ventricular ectopic rhythms

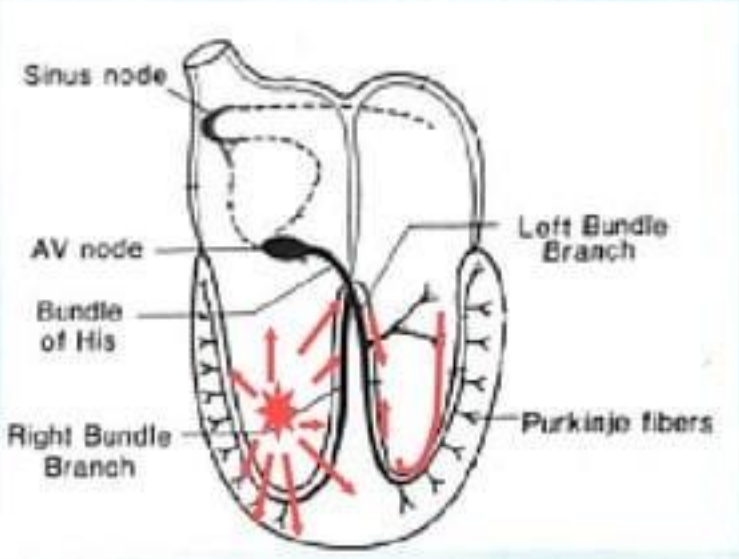
Cardiac arrhythmias

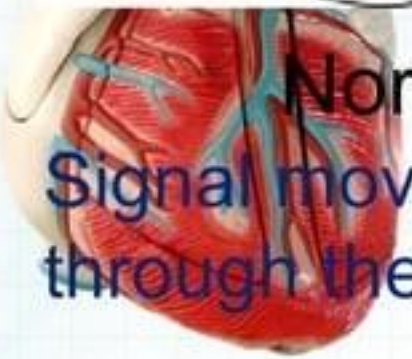


- Ventricular ectopic beats
- Ventricular tachycardia
- Ventricular fibrillation

Ventricular Conduction







Normal

Signal moves rapidly through the ventricles

Abnormal

Signal moves slowly through the ventricles

Cardiac arrhythmias

Ventricular ectopic beats

- The pt C/O extra beats, missed beats or heavy beats because it may be the premature beat, the post-ectopic pause or the next sinus beat that is noticed by the pt. .the pulse is irregular
- When a premature beat occurs regularly after every normal beat, 'pulsus bigeminus' may occur.

Cardiac arrhythmias

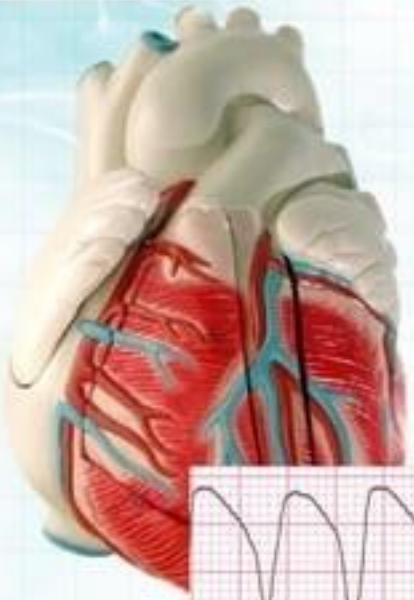
- in normal heart they may be found often prominent at rest & disappear with exercise. No treatment is required. Just ß-blocker to reduce palpitation and anxiety.
- However persistent frequent PVCs (>10/hr) indicate poor prognosis.

Ventricular tachycardia

Cardiac arrhythmias

- Ventricular tachycardia is defined as three or more consecutive PVCs. The usual rate is 160-240/min with regular rhythm.
- Ventricular tachycardia is either non-sustained (lasting < 30sec) or sustained.
- Pt may C/O Palpitation, dyspnea, dizziness or syncope.

Cardiac arrhythmias



CUASES

- Acute MI
- Myocarditis
- Dilated cardiomyopathy
- Hypertrohic cardiomyopathy
- Chronic ischemic disease
- Mitral valve prolapse

ECG;

ECG show a rapid ventricular rhythm with broad abnormal QRS complexes.

Cardiac arrhythmias

Torsade de pointes

- It is a form of ventricular tachycardia in which QRS morphology twists around baseline. May occur spontaneously in prolonged QT interval due to hypokalemia.

Treatment

Cardiac arrhythmias

Treatment may be urgent, depending on the haemodynamic situation. If the patient is haemodynamically compromised (e.g. hypotensive or pulmonary oedema) emergency DC cardioversion may be required. On the other hand, if the blood pressure and cardiac output are well maintained first-line drug treatment consists of lidocaine (50-100 mg i.v. over 5 minutes) followed by a lidocaine infusion (2-4 mg i.v. per minute).

Cardiac arrhythmias

if VT not suppressed by lidocaine then give I.V amiodarone (150mg over 10min followed by 360mg over 6hrs then 540mg over18hrs followed by 20-80mg/kg/min infusion). DC cardioversion is necessary if medical therapy is unsuccessful.

Cardiac arrhythmias

Chronic recurrent VT

Sustained VT

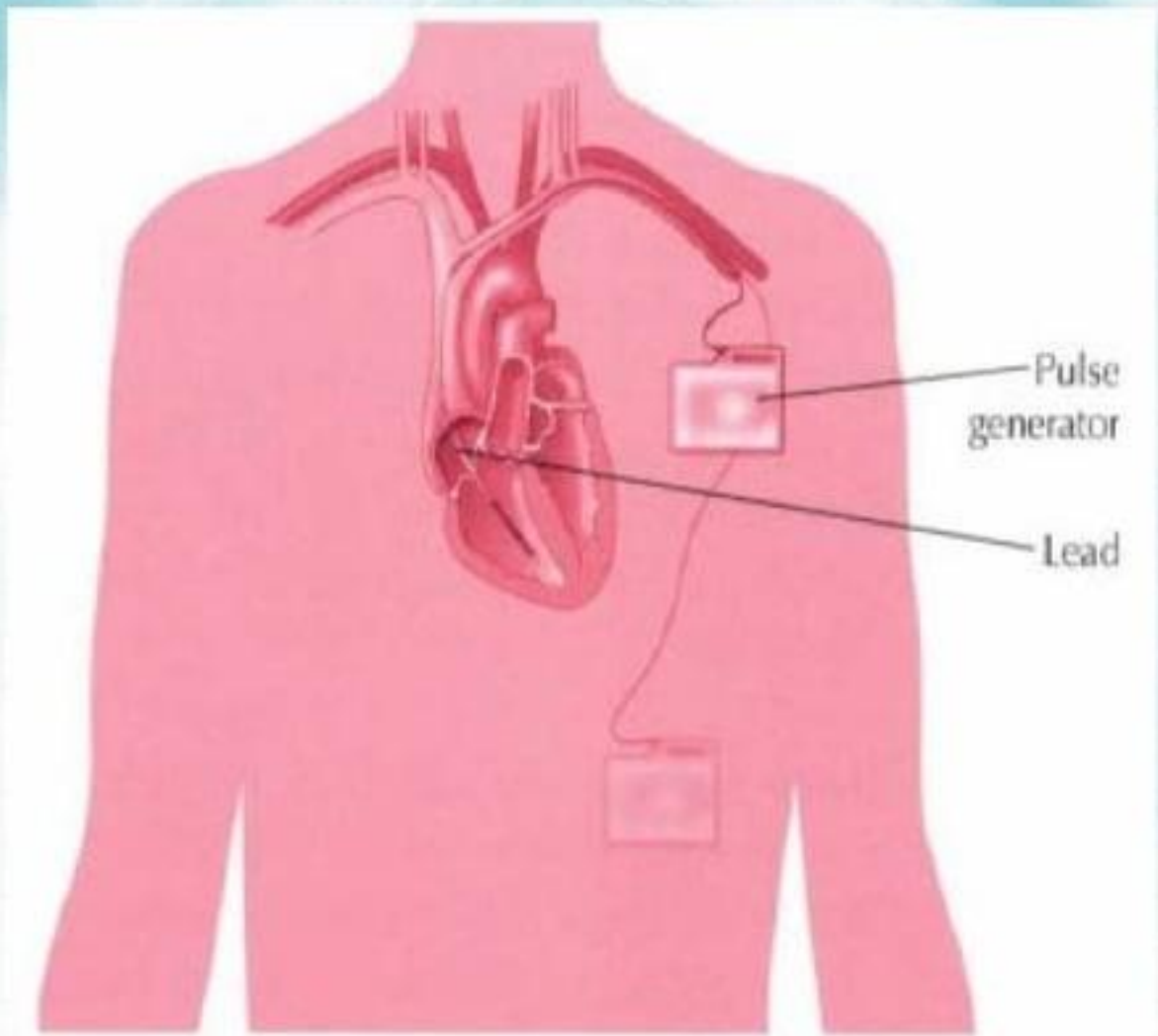
Treatment of choice is implantable cardioverter-defibrillator device (ICD).

Non sustained VT

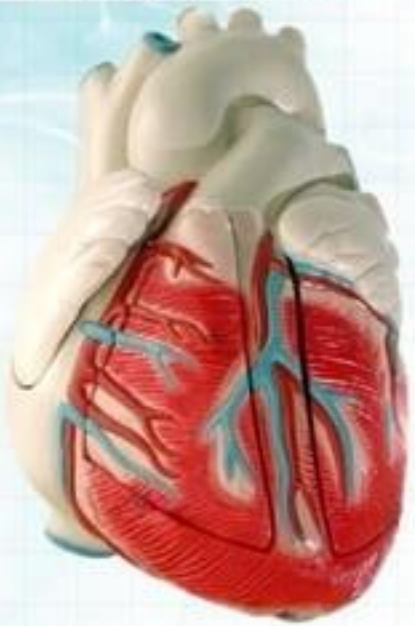
- Beta blockers reduce the incidence of sudden death by 40-50.
- amiodarone may be beneficial.
- implantable cardioverter-defibrillator device

Implantable defibrillator

Cardiac arrhythmias



Cardiac arrhythmias



CARDIAC ARREST

Cardiac arrest is sudden and complete loss of cardiac function. it may be due to:-

- Ventricular fibrillation
- Asystole

Ventricular fibrillation

Cardiac arrhythmias

This is very rapid and ineffective ventricular activation which produce no pulse. Therefore pt is pulseless and rapidly becomes unconscious and respiration ceases.

Causes:-

- MI
- Electric shock
- Hypokalemia
- Electrocution

Cardiac arrhythmias

Clinical features:-

- Loss of consciousness within seconds
- Pulse absent
- Respiration ceases.

ECG

shows shapeless oscillations

Management

- Electrical defibrillation. If it not available then perform CPR.
- In survivors of VT if cause is not revrsible, then ICD is 1st line therapy to manage further episodes.

Automatic external defibrillator

VENTRICULAR ASYSTOLE

Cardiac arrhythmias

- in this condition there is no electrical activity of the ventricles. It may be due to localized ventricular damage complicating MI.
- Treatment is cardiopulmonary resuscitation CPR.

WOLFF-PARKISON-WHITE SYNDROME

Cardiac arrhythmias

- Is a congenital condition caused by an abnormal myocardial connection between atrium & ventricle. In normal sinus rhythm conduction takes place through the AV node but in this condition it may bypass the AV node and conduct quickly over the abnormal connection. To depolarize the ventricle abnormally.

Cardiac arrhythmias

- because the bypass lacks the rate-limiting properties of the normal AV node, the pt are at risk of ventricular fibrillation.
- ECG Shows
- Short OR interval.
- Wide QRS complex.
- Delta wave: slurring of the beginning QRS complex.

Cardiac arrhythmias

- In these pts atrial premature contractions have 2 pathways to enter the ventricles
- AV node (normal) produce SVT with narrow QRS complex.
- Accessory pathway produce SVT with wide QRS complex.

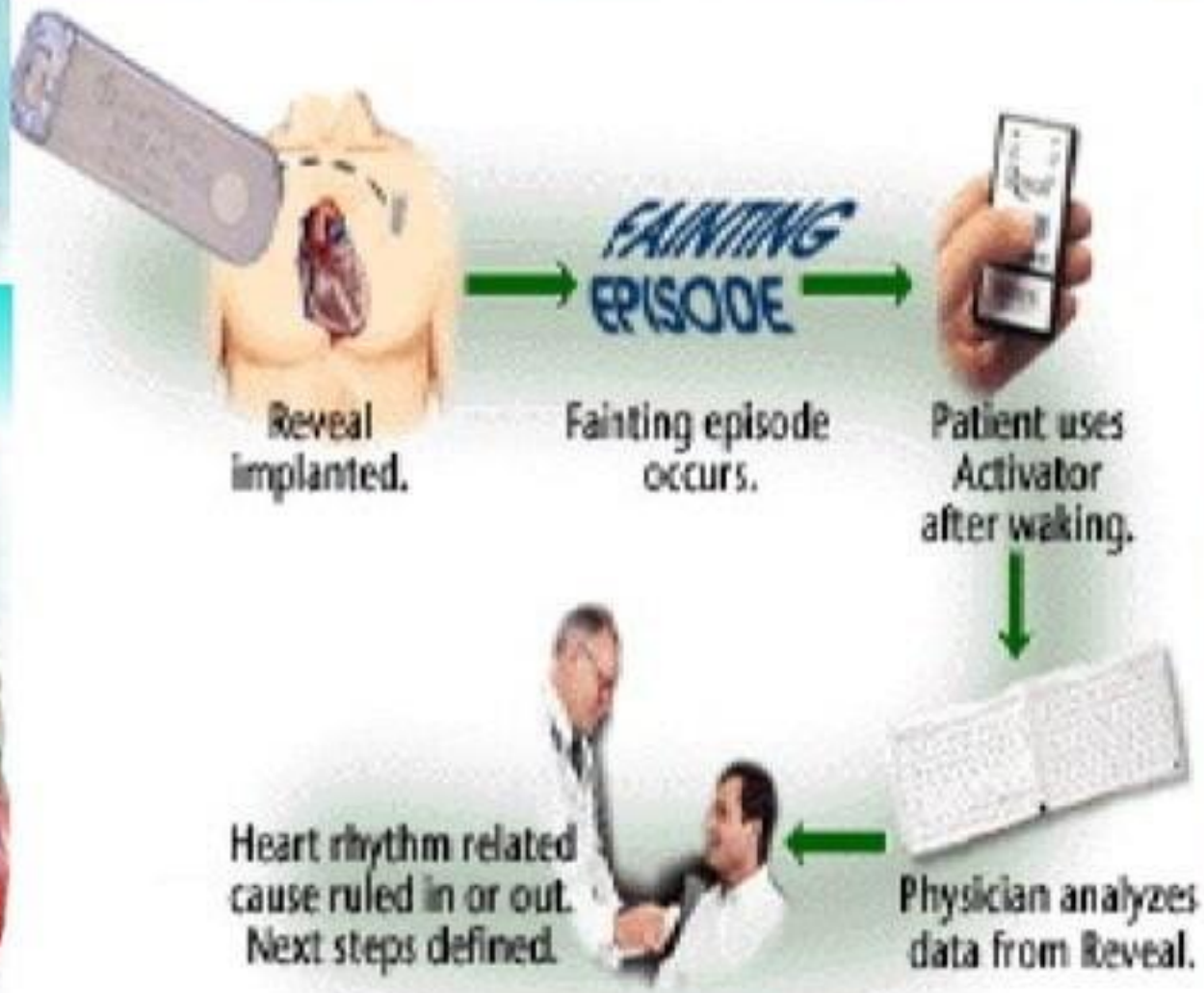
Cardiac arrhythmias

Treatment

- Asymptomatic pt require no treatment.
- unstable pt DC cardioversion
- Stable pt
  - SVT with narrow QRS complex adenosine or Verapamil.
  - SVT with wide QRS complex IV procainamide.

Medtronic implantable loop recorder

Cardiac arrhythmias


FAINTING
EPISODE
Reveal implanted.
Fainting episode occurs.
Patient uses Activator after waking.
Physician analyzes data from Reveal.
Heart rhythm related cause ruled in or out. Next steps defined.

Cardiac arrhythmias

The Reveal Insertable Loop Recorder continuously monitors the rate and rhythm of the heart. It works much like a black box in an airplane, whereby vital information is recorded during the actual fainting episode and can be played back later for detailed analysis. The Reveal Insertable Loop Recorder can continuously record the heart's rate and rhythm for up to 14 months.

© http://www.medtronic.com/reveal/new.html

THANK YOU